JACC Vol. 29, No. 1
January 1997:113–21

Long-Term Follow-Up After Radiofrequency Modification of the Atrioventricular Node in Patients With Atrial Fibrillation

FRED MORADY, MD, FACC, CAROL HASSE, RN, S. ADAM STRICKBERGER, MD, K. CHING MAN, DO, EMILE DAOUD, MD, FRANK BOGUN, RAJIVA GOYAL, MD, MARK HARVEY, MD, BRADLEY P. KNIGHT, MD, RAUL WEISS, MD, MARWAN BAHU, MD

Ann Arbor, Michigan

Objectives. **The purpose of this study was to describe the long-term follow-up results in 62 patients with atrial fibrillation and an uncontrolled ventricular rate, who underwent radiofrequency modification of the atrioventricular (AV) node.**

Background. **Previous studies in small numbers of patients have suggested that radiofrequency modification may be effective in controlling the ventricular rate in patients with atrial fibrillation, but long-term follow-up data have been lacking.**

Methods. **The subjects of this study were 62 consecutive patients (mean age ± SD 65 ± 14 years; 43 with structural heart disease) who underwent an attempt at radiofrequency modification of the AV node because of symptomatic, drug-refractory atrial fibrillation with an uncontrolled ventricular rate. The atrial fibrillation was chronic in 46 patients and paroxysmal in 16. Radiofrequency energy was applied to the posteroseptal or midseptal right atrium to lower the ventricular rate in atrial fibrillation to 120 to 130 beats/min during an infusion of 4 μg/min of isoproterenol.**

Results. **Short-term control of the ventricular rate was successfully achieved without the induction of pathologic AV block in 50 (81%) of 62 patients. Inadvertent high degree AV block occurred in 10 (16%) of 62 patients, with the AV block occurring at the time of the procedure in 6 patients and 36 to 72 h after the procedure in 4. During 19 ± 8 months of follow-up (range 4 to 33), 5 (10%) of 50 patients had a symptomatic recurrence of an uncontrolled rate during atrial fibrillation. Overall, adequate rate control at rest and during exertion, without pathologic AV block, was achieved long term in 45 (73%) of 62 patients. Among 37 patients with a successful outcome, left ventricular ejection fraction increased from (mean ± SD) 0.44 ± 0.14 to 0.51 ± 0.10 one year later ($p < 0.001$). Complications other than AV block included polymorphic ventricular tachycardia 10 to 24 h after the procedure in two patients who had a predisposing factor for ventricular tachycardia and sudden death 1 to 5 months after the procedure in two patients with idiopathic dilated cardiomyopathy, one of whom had a pacemaker for AV block.**

Conclusions. **In ~70% of properly selected patients with atrial fibrillation and an uncontrolled ventricular rate, radiofrequency modification of the AV node results in excellent long-term control of the ventricular rate at rest and during exertion.**

(J Am Coll Cardiol 1997;27:113–21)

Several studies (1–6) have demonstrated that it is feasible to modify atrioventricular (AV) node conduction without creating pathologic AV block in patients with atrial fibrillation and an uncontrolled ventricular rate using radiofrequency energy. However, the number of patients in these studies has been small, and the duration of follow-up has been relatively short. Several important issues have remained unclear, including the long-term changes in ventricular rate, the incidence of recurrence of rapid AV conduction, and the long-term effects on left ventricular function. The purpose of this study was to clarify these issues by describing the long-term follow-up in 62 patients with atrial fibrillation and an uncontrolled ventricular rate who underwent radiofrequency modification of the AV node to control the ventricular rate.

Methods

Patients. The subjects of this study were 62 consecutive patients who underwent an attempt at radiofrequency modification of the AV node at the University of Michigan Medical Center because of symptomatic, drug-refractory atrial fibrillation with an uncontrolled ventricular rate. In selecting patients to undergo this procedure, there were two exclusion criteria: 1) symptomatic bradycardia or pauses ≥2 s, not attributable to pharmacologic therapy; 2) the presence of symptoms at rest, such as those due to irregularity, in association with a ventricular rate during atrial fibrillation ≤90 beats/min. If one of these exclusion criteria was present, the patient underwent radiofrequency ablation of the AV junction and insertion of a permanent pacemaker. During the time that the 62 patients in this study were recruited, 17 other patients underwent ablation

From the Department of the Internal Medicine, Division of Cardiology, The University of Michigan Medical Center, Ann Arbor, Michigan.

Manuscript received April 8, 1996; revised manuscript received August 26, 1996, accepted September 20, 1996.

Address for correspondence: Dr. Fred Morady, University of Michigan Medical Center, 1500 East Medical Center Drive, B1F245, Ann Arbor, Michigan 48109-0022.

0735-1097/97/$17.00
PII S0735-1097(96)00445-7

JACC Vol. 29, No. 1
January 1997:113–21

Abbreviations and Acronyms

AH = atrial–His interval
AV = atrioventricular
ECG = electrocardiographic
HV = His–ventricular interval

of the AV node because of the presence of one of these exclusion criteria.

The clinical characteristics of the patient population are described in Table 1. Atrial fibrillation was chronic in 46 patients and paroxysmal in 16. Mean left ventricular ejection fraction determined by echocardiography or radionuclide ventriculography was 0.44 ± 0.14 (mean ± SD). Before the electrophysiologic procedure, a mean of 4.5 ± 1.3 antiarrhythmic drugs had been ineffective in maintaining sinus rhythm or controlling ventricular rate, including amiodarone in 24 patients. Every patient had symptoms of either palpitations or decreased exercise tolerance, or both. Mean Canadian Cardiovascular Society functional class before the electrophysiologic procedure was 3.0 ± 0.6. Twenty of the 62 patients were the subjects of two earlier reports and are included in the present series to provide long-term follow-up data (3,7).

Characterization of ventricular rate. As described previously (3), the ventricular rate was characterized under the following four conditions: 1) at rest, with the patient sitting; 2) after walking 50 yards at the patient's usual pace; 3) at peak exercise during a symptom-limited exercise treadmill test, except in patients in whom an exercise test was not feasible because of severe symptoms; 4) during 24-h ambulatory monitoring, to identify the slowest rate during atrial fibrillation. Whenever possible, these rates were measured in the absence of antiarrhythmic drug therapy, both before and after the radiofrequency procedure. However, in 15 patients who were being treated with digoxin, a beta-adrenergic antagonist or a calcium channel antagonist for reasons other than rate control, therapy with these agents was not discontinued.

Table 1. Clinical Characteristics of 62 Patients

Age (yr)	65 ± 14
Range	34–86
Men/women	32/30
Type of atrial fibrillation	
Chronic	46
Paroxysmal	16
Duration of symptoms (yr)	5.4 ± 5.6
Type of heart disease	
Coronary artery disease	16
Valvular	12
Idiopathic dilated cardiomyopathy	7
Hypertension	5
Hypertrophic cardiomyopathy	3
None	19
Left ventricular ejection fraction	0.44 ± 0.14

Data are expressed as mean value ± SD or number of patients, unless otherwise indicated.

Radiofrequency modification protocol. The study protocol was approved by the Human Research Committee at the University of Michigan. The technique has been described previously (3). After written informed consent was obtained, two quadripolar electrode catheters were positioned at the His bundle and in the right ventricle using a femoral vein approach. The catheter used to deliver radiofrequency energy was a 7F quadripolar electrode catheter with a 4-mm distal electrode, 2–5–2-mm interelectrode spacing and a deflectable tip (Mansfield Scientific or EP Technologies). Whenever necessary, a 60-cm guiding sheath (Daig Corp) was used to improve stability of the ablation catheter. Radiofrequency energy at a frequency of 500 kHz (EP Technologies) was delivered through the distal electrode of the ablation catheter. A large patch electrode (Valleylab) on the posterior thorax served as the indifferent electrode.

If the patient was in sinus rhythm at the onset of the procedure, the baseline atrial–His (AH) and His–ventricular (HV) intervals, the AV block cycle length and the AV node effective refractory period were measured. Effective refractory periods were determined at a basic drive cycle length of 600 ms. For the purpose of this study, if atrial refractoriness limited the determination of the AV node effective refractory period, the atrial effective refractory period was used as a substitute for the AV node effective refractory period.

If necessary, atrial fibrillation was induced by rapid atrial pacing. The ventricular rate during atrial fibrillation was measured in the baseline state and during infusion of isoproterenol at a rate of 4 μg/min. Applications of radiofrequency energy usually were delivered during the infusion of isoproterenol to allow immediate assessment of the effect of the radiofrequency energy under conditions simulating exercise.

The ablation catheter was positioned in the posteroseptal right atrium, at the level of the coronary sinus ostium, where the AV electrogram ratio was ≤0.5. Radiofrequency energy was delivered at an initial power setting of 12 W and slowly titrated upward to a maximum of 50 W for 1 min, according to temperature or impedance monitoring (8) and the response of the ventricular rate. The application of radiofrequency energy was immediately stopped whenever the RR interval abruptly lengthened. If there was no change in the ventricular rate, the catheter was positioned at progressively more superior and anterior positions along the right atrial septum. No applications were delivered at sites where the His bundle amplitude exceeded 0.02 mV (3). The end point of the procedure was a ventricular rate of 120 to 130 beats/min during the infusion of isoproterenol.

In eight patients who were in sinus rhythm at the onset of the procedure, electrical cardioversion was performed after completion of radiofrequency energy delivery, and the AH and HV intervals, AV block cycle length and AV node effective refractory period again were determined.

Follow-up evaluation. After the procedure, the patients underwent continuous electrocardiographic (ECG) monitor-

ing on an inpatient basis for at least 2 days and up to 4 days if transient high degree AV block had occurred during the procedure. A symptom-limited exercise treadmill test and 24-h ambulatory monitoring were performed 2 to 4 days after the procedure. The patients were seen in an outpatient clinic at intervals of 3, 12 and 24 months after the procedure, and a symptom-limited exercise treadmill test and 24-h ambulatory monitor recording were obtained at these times. Among the patients with paroxysmal atrial fibrillation, at least one episode of atrial fibrillation was documented by ambulatory monitoring between 4 and 24 months of follow-up. Left ventricular ejection fraction was measured by echocardiography or radionuclide ventriculography 12 months after the procedure. All patients have had at least 4 months of follow-up; 44 have had ≥12 months of follow-up; and 21 have had at least 24 months of follow-up.

Statistical analysis. Continuous variables are expressed as mean value ± SD and were compared using a paired *t* test or by analysis of variance with repeated measures. Multiple comparisons were performed using the Fisher least significant difference technique. Categoric variables were compared by contingency table analysis; $p < 0.05$ was considered significant.

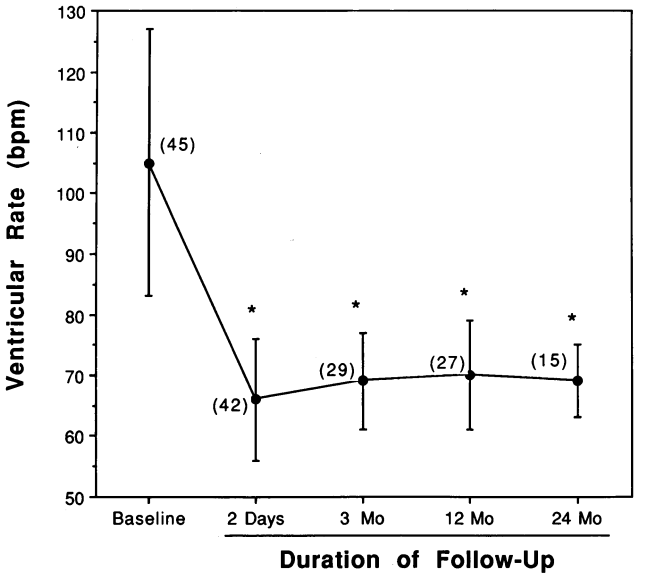

Figure 1. Mean ventricular rate at rest and while awake during atrial fibrillation, before and after radiofrequency modification of the AV node. bpm = beats/minute. **Vertical bars** = SD; **numbers in parentheses** = number of patients in whom heart rate data were available. *$p < 0.001$, significant difference versus baseline.

Results

Effective control of ventricular rate. At the time of discharge from the hospital 2 to 4 days after the radiofrequency procedure, the ventricular rate was successfully controlled without induction of pathologic AV block in 50 (81%) of the 62 patients. The mean number of applications of radiofrequency energy was 12 ± 8 (range 1 to 35), and the mean duration of fluoroscopy was 31 ± 17 min (range 8 to 75). The location of the radiofrequency energy application that resulted in slowing of the ventricular rate was posteroseptal in 15 patients and midseptal in 35.

During a mean follow-up period of 19 ± 8 months (range 4 to 33), 5 (10%) of the 50 patients had a symptomatic recurrence of an uncontrolled rate during atrial fibrillation; the symptoms recurred in these patients between 2 and 9 weeks after the procedure. Two of the five patients underwent a second attempt at radiofrequency modification of the AV node, which was effective long term. There were no identifiable predictors of recurrence of a rapid rate after an initially successful procedure.

Another 2 of the 50 patients, who underwent the procedure because of paroxysmal atrial fibrillation, had a recurrence of symptoms of palpitations and exercise intolerance 2 to 4 weeks afterward and were found to have paroxysmal atrial flutter as the cause of their symptoms. In these two patients, the ventricular rate at rest during atrial fibrillation was 64 to 76 beats/min compared with 110 to 130 beats/min at rest during atrial flutter.

Overall, adequate rate control without pathologic AV block was achieved long term in 45 (73%) of 62 patients. The long-term success rate was 76% among the 46 patients with chronic atrial fibrillation compared with 62% among the 16 patients with paroxysmal atrial fibrillation ($p = 0.3$).

Atrioventricular block. Inadvertent high degree AV block occurred in 10 (16%) of the 62 patients, with the AV block occurring at the time of the procedure in 6 patients and 36 to 72 h after the procedure in 4. The application of radiofrequency energy that resulted in high degree AV block was posteroseptal in three patients and midseptal in seven. A His bundle depolarization was absent at eight of these sites and present but <0.02 mV in amplitude at two of the sites. Each of the four patients in whom the onset of high degree AV block was delayed had transient third-degree AV block lasting 10 to 27 s during the initial procedure. Transient third-degree AV block also occurred during the procedure in 16 patients who had a successful outcome.

Each of the 10 patients who developed inadvertent high degree AV block had a junctional escape rhythm at a rate of 40 to 58 beats/min and underwent implantation of a permanent pacemaker.

Ablation of the AV junction and pacemaker implantation were performed intentionally in 7 (11%) of the 62 patients: at the time of the initial procedure in 2; after a recurrence of a rapid ventricular rate during atrial fibrillation at 2 to 9 weeks of follow-up in 3; and for episodes of paroxysmal atrial flutter that began at 2 to 4 weeks of follow-up in 2.

Ventricular rate. The mean ventricular rates during atrial fibrillation, at rest and during ambulation, measured before the radiofrequency modification procedure and at 2 days and at 3, 12 and 24 months after the procedure in the 45 patients who had a successful outcome are shown in Figures 1 and 2. The mean maximal ventricular rates during exercise before the

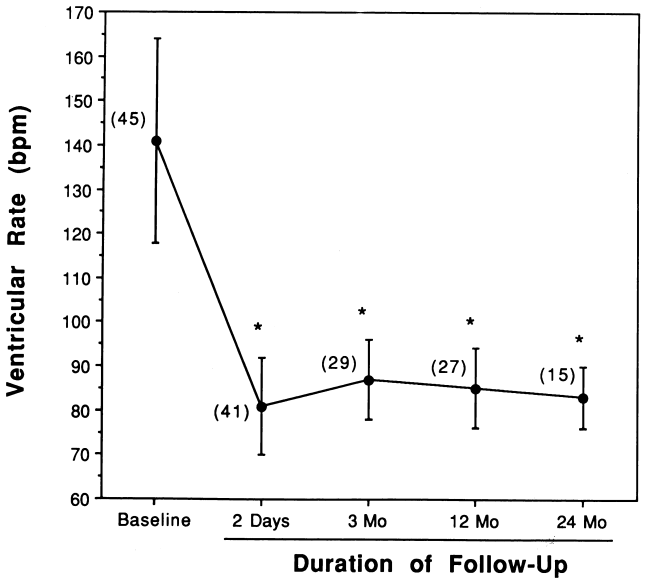

Figure 2. Mean ventricular rate during atrial fibrillation after walking 50 yards, before and after radiofrequency modification of the AV node. bpm = beats/minute. Format as in Figure 1. *p < 0.001, significant difference versus baseline.

procedure and at 2 days and 3, 12 and 24 months of follow-up are shown in Figure 3. For each of the conditions under which the ventricular rate was measured, there was a significant decrease compared with baseline values throughout the period of follow-up. There were no significant changes in rate at 2 days and 24 months of follow-up, except in the case of the

Figure 3. Mean ventricular rate during atrial fibrillation at peak exercise on a treadmill, before and after radiofrequency modification of the AV node. bpm = beats/minute. Format as in Figure 1. *p < 0.001, significant difference versus baseline. #p < 0.05, significant change at 12 and 24 months versus 2 days of follow-up.

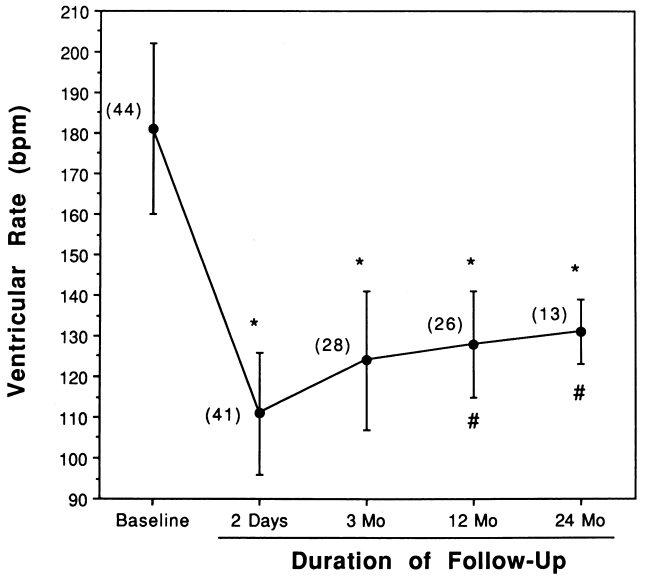

Table 2. Atrioventricular Node Properties and His–Ventricular Interval Before and After Radiofrequency Modification in Eight Patients

	Before RF Modification	After RF Modification	p Value
Sinus cycle length (ms)	742 ± 98	732 ± 90	0.1
Atrial–His interval (ms)	86 ± 14	123 ± 28	< 0.01
His–ventricular interval (ms)	45 ± 5	45 ± 5	1.0
AVN block cycle length (ms)	335 ± 28	432 ± 64	< 0.01
AVN ERP (ms)	275 ± 29	369 ± 70	< 0.01
Dual AVN physiology	2/8 (25%)	0/8	0.4

Data are expressed as mean value ± SD or number (%) of patients. AVN = atrioventricular node; ERP = effective refractory period; RF = radiofrequency.

maximal rate, which was significantly greater at 12 and 24 months than at 2 days of follow-up.

Atrioventricular node properties. Atrioventricular node properties were measured before and after a successful radiofrequency modification procedure in eight patients. The mean AH interval, AV block cycle length and AV node effective refractory period were significantly prolonged by the procedure (Table 2). Dual-AV node physiology was present in two patients before the procedure and in no patient afterward.

Symptoms and functional class. Among the 45 patients in whom the ventricular rate was controlled long term after the radiofrequency modification procedure, 33 (82%) of 40 with a history of palpitations reported that the palpitations had resolved or were infrequent and mild, and the remaining 7 patients (18%) described the palpitations as improved and tolerable, but present every day. Among the 17 patients who underwent pacemaker implantation because of third-degree AV block, 13 (87%) of 15 with a history of palpitations reported resolution of symptoms during long-term follow-up, and the other 2 described persistent palpitations as mild. The percentage of patients who reported resolution of palpitations did not differ significantly between the two groups of patients (p = 0.7).

The mean Canadian Cardiovascular Society functional class improved from 3.0 ± 0.5 to 1.6 ± 0.5 (p < 0.001) among the 45 patients with a successful outcome from the radiofrequency modification procedure. Among the 17 patients who received a pacemaker, the mean functional class improved from 3.1 ± 0.5 to 1.5 ± 0.4 (p < 0.001). The degree of improvement in functional class did not differ significantly between the patients in whom the radiofrequency modification procedure was successful and those who received a pacemaker (p = 0.9).

Complications other than AV block. Two patients (3%) had documented episodes of polymorphic ventricular tachycardia 10 to 24 h after the radiofrequency modification procedure. One patient, a 69-year old woman with dilated, nonischemic cardiomyopathy who was the subject of an earlier report (7), developed sustained polymorphic ventricular tachycardia as a complication of amiodarone therapy 5 days before the modification procedure and had a recurrence 10 h after the procedure, when the average ventricular rate during atrial

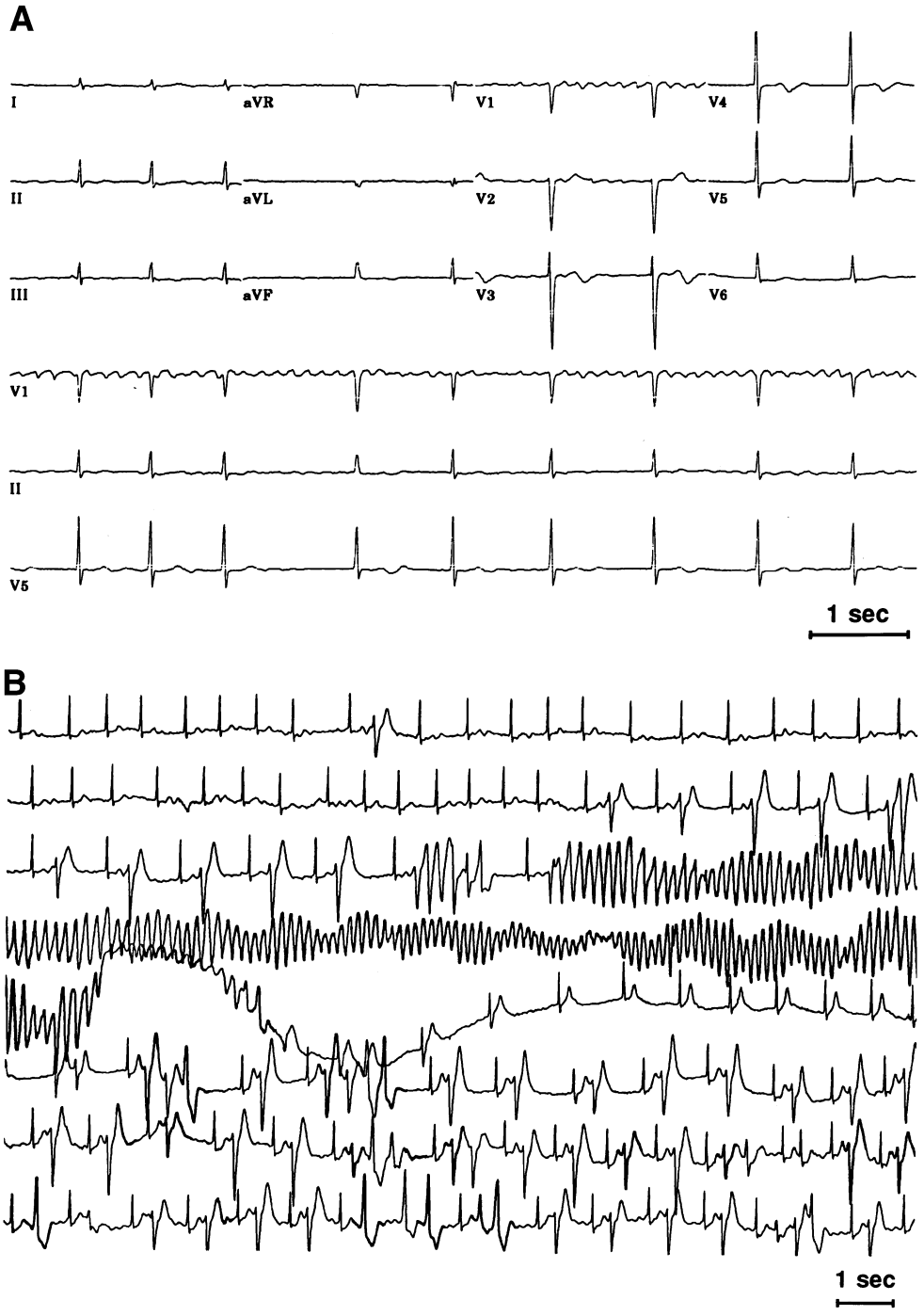

Figure 4. Example of polymorphic ventricular tachycardia (VT) after radiofrequency modification in a patient who had a predisposing factor for torsade de pointes. **A,** ECG recorded 18 h after the modification procedure. The average ventricular rate is 56 beats/min. The corrected QT interval was 560 ms. The QT prolongation was not recognized. **B,** Twenty-four hours after the procedure, the patient developed ventricular bigeminy and a 30-s episode of polymorphic VT consistent with torsade de pointes. The VT had a cycle length of 170 to 200 ms and was accompanied by transient loss of consciousness. The serum potassium concentration measured immediately after the episode of VT was 3.8 mEq/liter. Polymorphic VT did not recur after administration of potassium chloride and the institution of ventricular pacing at a rate of 85 beats/min.

fibrillation was 70 beats/min and the corrected QT interval was 570 ms. The other patient was a 62-year old woman with moderately severe aortic and mitral stenosis and a left ventricular ejection fraction of 0.50 who experienced syncope in association with a 30-s episode of polymorphic ventricular tachycardia 24 h after the procedure, when the average ventricular rate during atrial fibrillation was 70 beats/min and the corrected QT interval was 560 ms (Fig. 4). The serum potassium concentration was 3.8 mEq/liter shortly after the episode of ventricular tachycardia. In both patients, a pacemaker was implanted, and ventricular tachycardia did not recur during 3 months of pacing at a rate of 80 to 90 beats/min. The pacing rate was then lowered to 60 beats/min, with no episodes of symptomatic ventricular tachycardia during 11 to 22 months of follow-up.

Two other patients (3%) died suddenly 1 to 5 months after the procedure. Both were men, 52 and 71 years old, and both had idiopathic dilated cardiomyopathy and a left ventricular ejection fraction of 0.20. One patient had developed third-degree AV block as a complication of the modification proce-

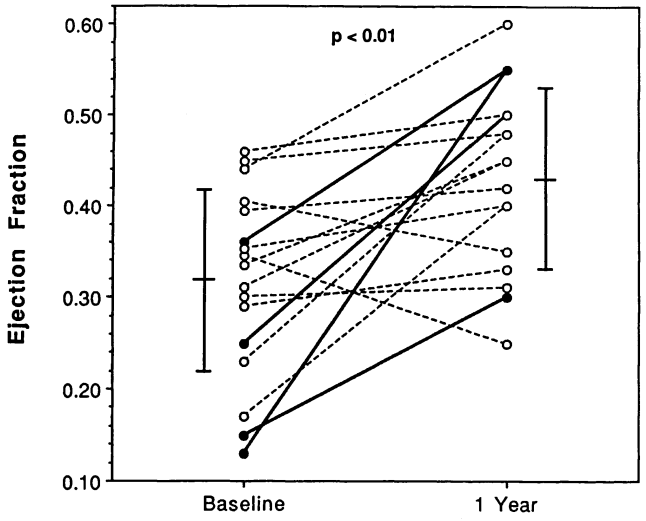

Figure 5. Left ventricular ejection fraction before and 1 year after radiofrequency modification of the AV node in 17 patients who had a successful outcome and in whom the baseline ejection fraction was <0.50. **Solid lines** = four patients with idiopathic dilated cardiomyopathy; **dashed lines** = patients with other types of heart disease. Also shown is the mean ± SD at baseline and at 1 year of follow-up ($p < 0.001$).

dure, underwent pacemaker implantation, was discharged from the hospital with the pacemaker programmed in a VVIR mode with rates of 70 to 120 beats/min and died suddenly 1 month later. In the other patient, the ventricular rate during atrial fibrillation was successfully controlled by the radiofrequency modification procedure, but the patient died suddenly 5 months later. Neither patient underwent a postmortem examination.

Two patients died of preexisting end-stage pulmonary hypertension or chronic renal failure 1 to 2 weeks after the radiofrequency modification procedure.

Left ventricular function. Among 50 patients in whom left ventricular ejection fraction was measured before and 1 year after the radiofrequency modification procedure, mean ejection fraction increased from 0.45 ± 0.13 to 0.50 ± 0.11 ($p = 0.001$). In 37 of these patients who had a successful outcome, left ventricular ejection fraction increased from 0.44 ± 0.14 to 0.51 ± 0.10 ($p < 0.001$), and in 13 patients with third-degree AV block and a pacemaker, the baseline mean ejection fraction of 0.47 ± 0.12 remained unchanged at 0.46 ± 0.13 ($p = 0.3$).

Within the group of patients with a successful outcome, there were 17 patients in whom baseline left ventricular ejection fraction was <0.50 and in whom a repeat determination of ejection fraction was available 1 year after the procedure. The mean ejection fraction of 0.32 ± 0.10 in this subgroup of patients increased significantly to 0.43 ± 0.10 ($p < 0.01$) (Fig. 5). Among the seven patients in this study who had dilated nonischemic cardiomyopathy, measurement of left ventricular ejection fraction at baseline and 1 year after the modification procedure was available in four; in these four patients, mean ejection fraction increased from 0.22 ± 0.10 to 0.48 ± 0.12 ($p = 0.02$) (Fig. 5).

Among the patients who required a pacemaker, there were six patients in whom baseline left ventricular ejection fraction was <0.50 and in whom a repeat determination of ejection fraction was available 1 year after the procedure. Baseline ejection fraction in these patients was 0.33 ± 0.06 before ablation and 0.33 ± 0.08 1 year after ablation ($p = 0.7$).

Discussion

Main findings. The results of this study indicate that in patients with atrial fibrillation and a rapid ventricular rate, adequate rate control without the need for a permanent pacemaker can be achieved long term in ~70% of patients. Although the short-term success rate was ~80%, there was a 10% recurrence rate of rapid AV conduction in the first 3 months after the procedure. An important limitation of the radiofrequency modification technique used in this study was a 16% incidence of unintentional third-degree AV block. Other potential complications included polymorphic ventricular tachycardia in patients who already had a predisposing factor for polymorphic ventricular tachycardia and possibly sudden death.

Among the patients who did not have recurrence of an uncontrolled ventricular rate within the first 3 months after the procedure, the ventricular rates during atrial fibrillation at rest and during mild exertion remained stable for up to 2 years of follow-up. Although the maximal ventricular rate during exercise increased mildly after 3 months of follow-up, a highly significant reduction in peak rate relative to baseline was maintained for up to 2 years. The significant improvement in symptoms and functional class that accompanied the long-term control of ventricular rate was comparable in magnitude to the symptomatic improvement that occurs after AV junction ablation and pacemaker implantation. In some patients, long-term control of the ventricular rate was accompanied by an increase in left ventricular ejection fraction, and the improvement in left ventricular function was sometimes dramatic, particularly in patients with dilated cardiomyopathy that was apparently idiopathic.

Atrioventricular block. The incidence of high degree AV block occurring as a complication of the radiofrequency modification procedure was 16%. Because all the patients in this study also were appropriate candidates for AV node ablation, and because the clinical response to AV junction ablation and pacemaker implantation in patients with atrial fibrillation and an uncontrolled ventricular rate is usually excellent, a relatively high risk of AV block did not serve as a deterrent to the modification procedure. However, the 16% incidence of AV block indicates that an attempt at radiofrequency modification may be inappropriate in patients who are not symptomatic enough to warrant AV node ablation or in patients who find the possibility of pacemaker implantation unacceptable.

Almost 50% of the cases of inadvertent AV block occurred on a delayed basis, 36 to 72 h after the procedure. As was the

case in an earlier report (3), the occurrence of transient high degree AV block during the radiofrequency modification procedure was a predictor of delayed, persistent AV block, and the delayed onset of AV block was never observed in patients who did not develop transient AV block during the procedure. Therefore, although it is possible that the procedure could be performed safely on an outpatient basis in some patients, up to 72 h of inpatient ECG monitoring is appropriate if transient AV block occurs during the radiofrequency modification procedure.

Relief of symptoms. Ablation of the AV node and insertion of a permanent pacemaker usually results in a major improvement in symptoms related to atrial fibrillation and an uncontrolled ventricular rate (9,10). This has been found in previous studies and was also the case among the patients in the present study who required a pacemaker after AV block was induced either unintentionally or intentionally. Whereas ablation and pacemaker implantation result in both control and regularization of the ventricular rate, the ventricular rate is controlled but still irregular after the modification procedure. Nevertheless, the patients in this study who had a successful outcome from the standpoint of rate control also had an excellent clinical response, with as much improvement in symptoms as the patients who required a pacemaker. However, an attempt was made to screen out patients whose symptoms were attributable to irregularity and not rapidity of the ventricular rate.

Left ventricular function. Tachycardia-induced cardiomyopathy is a well recognized entity that has been demonstrated to be at least partially reversible after radiofrequency ablation of incessant tachycardia or after control of the ventricular rate by either pharmacologic therapy or by AV node ablation and pacemaker insertion in patients with atrial fibrillation (11–21). The results of the present study demonstrate that significant improvement in left ventricular ejection fraction also may occur after radiofrequency modification of the AV node in patients with atrial fibrillation. Although an improvement in left ventricular ejection fraction was not limited to patients with a particular type of heart disease, the improvement was especially notable in patients with dilated cardiomyopathy. In some of these patients, a markedly abnormal left ventricular ejection fraction became normalized by 1 year after the modification procedure, suggesting that the idiopathic dilated cardiomyopathy was actually a reversible tachycardia-induced cardiomyopathy. However, the possibility that an improvement in ejection fraction was spontaneous or related to changes in drug therapy cannot be ruled out.

Left ventricular ejection fraction may improve after control of the ventricular rate in patients with atrial fibrillation because of either a change in loading conditions or intrinsic myocardial contractility. The simple measurements of left ventricular ejection fraction obtained in this study do not allow differentiation of whether the improvement was due only to a change in or was independent of loading conditions. A recent study (22) demonstrated that in young patients with chronic tachycardia, an improvement in myocardial function after ablation of the tachycardia is attributable to an increase in contractility, independent of cardiac loading conditions. Whether these findings apply to atrial fibrillation with an uncontrolled rate remains to be determined.

Polymorphic ventricular tachycardia. The incidence of polymorphic ventricular tachycardia after radiofrequency modification of the AV node in this study was 3%. In both patients who experienced this complication, there was an underlying predisposing factor, either amiodarone-induced proarrhythmia or hypokalemia (23,24). Both patients displayed QT prolongation before the episodes of ventricular tachycardia, and in both, the ventricular tachycardia was consistent with torsade de pointes. Because this type of arrhythmia may be suppressed by an increase in rate and facilitated by a decrease in rate (25,26), it is likely that the relative bradycardia that ensued after the radiofrequency modification procedure was an aggravating factor, even though the average rate of 70 beats/min at the time of polymorphic ventricular tachycardia was not excessively slow in absolute terms.

The occurrence of polymorphic ventricular tachycardia in the two patients in this study indicates that it may be appropriate to implant a permanent pacemaker after radiofrequency modification of the AV node, even in the absence of high degree AV block, in patients who have QT prolongation or a predisposition to polymorphic ventricular tachycardia. A pacemaker allows maintenance of a minimal rate of 80 to 90 beats/min, which may be sufficient to prevent episodes of torsade de pointes (26,27).

Sudden death. Two patients in this study died suddenly, one after an apparently successful radiofrequency modification procedure and the other after AV node ablation and pacemaker implantation. Both patients had nonischemic cardiomyopathy and markedly reduced left ventricular function, a condition known to be associated with sudden death (28). Whether sudden death in these patients was related to the radiofrequency procedure or was a result of the underlying heart disease is unclear. In the patient whose rate was successfully controlled by the modification procedure, the possibility of bradycardia-facilitated ventricular tachycardia cannot be ruled out. However, the 2% incidence of sudden death among the patients in this study who did not require a pacemaker compares favorably with the incidence of sudden death after radiofrequency ablation of AV conduction and implantation of a permanent pacemaker, which has been reported to be as high as 4% to 6% (29,30). Therefore, even if radiofrequency modification of the AV node does predispose to sudden death in some patients, the risk is no higher, and may be lower, than after AV node ablation and pacemaker implantation.

Comparison with previous studies. The long-term success rate of 73% in this study is similar to the 70% to 74% success rate reported in two earlier studies (3,4). However, in two other studies, the success rate of the radiofrequency modification procedure varied widely, from 25 to 85% (5,6). The number of patients in these previous studies was similar, ranging from 10 to 20 (3–6). Although the techniques used to modify AV conduction in those studies were similar in general terms, there were specific differences, including the rhythm at

the time of radiofrequency energy delivery, the use of atropine or isoproterenol during the procedure and the specific end points of the procedure. Because of these differences, comparisons between the success rates of these previous studies may not be meaningful.

Mechanism of rate control. Some previous studies (2,4,5,31) have suggested that control of the ventricular rate during atrial fibrillation results from ablation of the slow pathway or posterior inputs into the AV node. Two findings in the present study make this possibility unlikely: 1) In the several patients in whom AV node properties were measurable before and after the modification procedure, there was a consistent lengthening of the AH interval and AV block cycle length, effects that are not observed after slow pathway ablation (32). 2) The incidence of AV block in this study was 16% compared with a <2% incidence of high degree AV block after slow pathway ablation (32–36). As previously indicated (3), these findings are consistent with direct effects on the compact AV node rather than simply the posterior inputs to the AV node.

Study limitations. A limitation of this study is that patients were not randomly assigned to undergo modification or ablation of the AV node. Therefore, no definitive conclusions are possible regarding comparisons of the two procedures relative to relief of symptoms, improvement in functional class, effects on left ventricular function or complication rates. A second limitation is that quality of life was not assessed in a prospective, systematic fashion using a standardized measurement instrument.

Conclusions. In ~70% of properly selected patients with atrial fibrillation and an uncontrolled ventricular rate, radiofrequency modification of the AV node results in excellent long-term control of the ventricular rate at rest and during exertion, relief of symptoms and improvement in functional class. Furthermore, in some patients with left ventricular dysfunction, particularly in patients with idiopathic dilated cardiomyopathy, slowing of the ventricular rate by the modification procedure may be associated with marked improvement in ejection fraction. Compared with radiofrequency ablation of the AV node and pacemaker insertion, the principal drawbacks of the modification procedure are a recurrence rate of 10% (vs. 0% to 5% after ablation [30,37]) and facilitation of polymorphic ventricular tachycardia in patients who are already predisposed to this arrhythmia. In contrast, an important advantage of the modification procedure is that, if successful, it obviates the need for a pacemaker, thereby eliminating the possibility of pacemaker-related complications and the short- and long-term costs incurred by lifetime pacemaker therapy.

Among patients with atrial fibrillation who require nonpharmacologic therapy, ablation of the AV node and pacemaker implantation may be more appropriate than AV node modification in patients whose symptoms of palpitations occur when the ventricular rate is controlled, in patients who may have a predisposition to polymorphic ventricular tachycardia and in patients who require or who already have a pacemaker because of bradyarrhythmia. In other patients with atrial fibrillation and a rapid ventricular rate, an attempt at radiofrequency modification of the AV node appears to be an appropriate alternative to complete ablation of AV conduction.

References

1. Kunze KP, Schluter M, Geiger M, Kuck KH. Modulation of atrioventricular nodal conduction using radiofrequency current. Am J Cardiol 1988;61: 657–8.
2. Fleck RP, Chen PS, Boyce K, Ross R, Dittrich HC, Feld GK. Radiofrequency modification of atrioventricular conduction by selective ablation of the low posterior septal right atrium in a patient with atrial fibrillation and a rapid ventricular response. Pacing Clin Electrophysiol 1993;16:377–81.
3. Williamson BD, Man KC, Daoud E, Niebauer M, Strickberger SA, Morady F. Radiofrequency catheter modification of atrioventricular conduction to control the ventricular rate during atrial fibrillation. N Engl J Med 1994;331: 910–7.
4. Feld GK, Fleck RP, Fujimura O, Prothro DL, Bahnson TD, Ibarra M. Control of rapid ventricular response by radiofrequency catheter modification of the atrioventricular node in patients with medically refractory atrial fibrillation. Circulation 1994;90:2299–307.
5. Bella PD, Carbucicchio C, Tondo C, Riva S. Modulation of atrioventricular conduction by ablation of the "slow" atrioventricular node pathway in patients with drug-refractory atrial fibrillation or flutter. J Am Coll Cardiol 1995;25:39–46.
6. Brignole M, Menozzi C, Gianfranchi L, et al. Single-session-step-wise radiofrequency catheter modification of atrioventricular conduction in patients with paroxysmal atrial fibrillation. Eur J Cardiovasc Pacing Electrophysiol 1995;5:18–22.
7. Williamson BD, Hummel J, Niebauer M, et al. Bradycardia-facilitated polymorphic ventricular tachycardia caused by amiodarone after radiofrequency modification of atrioventricular conduction. Am Heart J 1995;130: 399–401.
8. Strickberger SA, Ravi S, Daoud E, Niebauer M, Man KC, Morady F. Relation between impedance and temperature during radiofrequency ablation of accessory pathways. Am Heart J 1995;130:1026–30.
9. Rosenqvist M, Lee MA, Moulinier L, et al. Long-term follow-up of patients after transcatheter direct current ablation of the atrioventricular junction. J Am Coll Cardiol 1990;6:1467–74.
10. Brignole M, Gianfranchi L, Menozzi C, et al. Influence of atrioventricular junction radiofrequency ablation in patients with chronic atrial fibrillation and flutter on quality of life and cardiac performance. Am J Cardiol 1994;74:242–6.
11. Packer DL, Bardy GH, Worley SJ, et al. Tachycardia-induced cardiomyopathy: a reversible form of left ventricular dysfunction. Am J Cardiol 1986;57:563–70.
12. Lemery R, Brugada P, Cheriex E, Wellens HJ. Reversibility of tachycardia-induced left ventricular dysfunction after closed-chest catheter ablation of the atrioventricular junction for interactable atrial fibrillation. Am J Cardiol 1987;60:1406–8.
13. Peters KG, Kienzle MG. Severe cardiomyopathy due to chronic rapidly conducted atrial fibrillation: complete recovery after restoration of sinus rhythm. Am J Med 1988;85:242–4.
14. Cruz FE, Cheriex EC, Smeets JL, et al. Reversibility of tachycardia-induced cardiomyopathy after cure of incessant supraventricular tachycardia. J Am Coll Cardiol 1990;16:739–44.
15. Heinz G, Siostrzonek P, Kreiner G, Gossinger H. Improvement in left ventricular systolic function after successful radiofrequency His bundle ablation for drug refractory, chronic atrial fibrillation and recurrent atrial flutter. Am J Cardiol 1992;69:489–92.
16. Kieny JR, Sacrez A, Facello A, et al. Increase in radionuclide left ventricular ejection fraction after cardioversion of chronic atrial fibrillation in idiopathic dilated cardiomyopathy. Eur Heart J 1992;13:1290–5.
17. Grogan M, Smith HC, Gersh BJ, Wood DL. Left ventricular dysfunction due to atrial fibrillation in patients initially believed to have idiopathic dilated cardiomyopathy. Am J Cardiol 1992;69:1570–3.
18. Twidale N, Sutton K, Bartlett L, et al. Effects on cardiac performance of atrioventricular node catheter ablation using radiofrequency current for

drug-refractory atrial arrhythmias. Pacing Clin Electrophysiol 1993;16:1275–84.
19. Van Gelder IC, Crijns HJ, Blanksma PK, et al. Time course of hemodynamic changes and improvement of exercise tolerance after cardioversion of chronic atrial fibrillation unassociated with cardiac valve disease. Am J Cardiol 1993;72:560–6.
20. Rodriguez LM, Smeets JL, Xie B, et al. Improvement in left ventricular function by ablation of atrioventricular nodal conduction in selected patients with lone atrial fibrillation. Am J Cardiol 1993;72:1137–41.
21. Fenelon G, Wijns W, Andries E, Brugada P. Tachycardiomyopathy: mechanisms and clinical implications. Pacing Clin Electrophysiol 1996;19:95–106.
22. Fishberger SB, Colan SD, Saul P, Mayer JE, Walsh EP. Myocardial mechanics before and after ablation of chronic tachycardia. Pacing Clin Electrophysiol 1996;19:42–9.
23. Hohnloser SH, Klingenheben T, Singh BN. Amiodarone-associated proarrhythmic effects. Ann Intern Med 1994;121:529–35.
24. Kay GN, Plumb VJ, Arciniegas JG, Henthorn RW, Waldo AL. Torsade de pointes: the long-short initiating sequence and other clinical features: Observations in 32 patients. J Am Coll Cardiol 1983;5:806–17.
25. Jackman WM, Friday KJ, Anderson JL, Aliot EM, Clark M, Lazzara R. The long QT syndromes: a critical review, new clinical observations and a unifying hypothesis. Prog Cardiovasc Dis 1988;31:115–72.
26. Leenhardt A, Coumel P, Slama R. Torsades de pointes. J Cardiovasc Electrophysiol 1992;3:281–92.
27. Eldar M, Griffin JC, Abbott JA, et al. Permanent cardiac pacing in patients with the long QT syndrome. J Am Coll Cardiol 1987;10:600–7.
28. Akhtar M, Garan H, Lehmann MH, Troup PJ. Sudden cardiac death: management of high-risk patients. Ann Intern Med 1991;114:449–512.
29. Jackman WM, Wang X, Friday K, et al. Catheter ablation of atrioventricular junction using radiofrequency current in 17 patients: comparison of standard and large-tip catheter electrodes. Circulation 1991;83:1562–76.
30. Olgin JE, Scheinman MM. Comparison of high energy direct current and radiofrequency catheter ablation of the atrioventricular junction. J Am Coll Cardiol 1993;21:557–64.
31. Blanck Z, Dhala AA, Sra J, et al. Characterization of atrioventricular nodal behavior and ventricular response during atrial fibrillation before and after a selective slow-pathway ablation. Circulation 1995;91:1086–94.
32. Jackman WM, Beckman KJ, McClelland JH, et al. Treatment of supraventricular tachycardia due to atrioventricular nodal reentry by radiofrequency catheter ablation of slow pathway conduction. N Engl J Med 1992;327:313–8.
33. Jazayeri MR, Hempe SL, Sra JS, et al. Selective transcatheter ablation of the fast and slow pathways using radiofrequency energy in patients with atrioventricular nodal reentrant tachycardia. Circulation 1992;85:1318–28.
34. Kay GN, Epstein AE, Dailey SM, Plumb VJ. Selective radiofrequency ablation of the slow pathway for the treatment of atrioventricular nodal reentrant tachycardia. Evidence for involvement of perinodal myocardium within the reentrant circuit. Circulation 1992;85:1675–88.
35. Haissaguerre M, Gaita F, Fischer B, et al. Elimination of atrioventricular nodal reentrant tachycardia using discrete slow potentials to guide application of radiofrequency energy. Circulation 1992;85:2162–75.
36. Wathen M, Natale A, Wolfe K, Yee R, Newman D, Klein G. An anatomically guided approach to atrioventricular node slow pathway ablation. Am J Cardiol 1992;70:886–9.
37. Morady F, Calkins H, Langberg JJ, et al. A prospective randomized comparison of direct current and radiofrequency ablation of the atrioventricular junction. J Am Coll Cardiol 1993;21:102–9.